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*Original Research*

# Evaluation of the efficacy of high-dose n-acetylcysteine in preventing contrast-induced nephropathy in the emergency department



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**Abstract**

Aim: The aim of this study is to evaluate the efficacy of high-dose N-Acetylcysteine (NAC) in preventing contrast-induced nephropathy (CIN) in patients with mild renal dysfunction in the emergency department (ED), compared to normal saline (NS).

Material and Methods: This retrospective analysis is based on a previously conducted randomized controlled trial (RCT). The study includes a retrospective analysis of data from an RCT carried out in the ED at a tertiary hospital. A total of 48 patients were deemed eligible for analysis; 23 patients received NAC treatment, while 25 patients received saline treatment. Propensity score matching (PSM) was used to balance the baseline characteristics between the groups.

Results: After propensity score matching, 23 patients remained in each group. In the group treated with NAC in the ED, a significantly greater reduction in serum creatinine (SCr) levels was observed at 24 hours (-0.27 ± 0.15 mg/dL vs. -0.08 ± 0.29 mg/dL, p = 0.022) and 72 hours (-0.46 ± 0.22 mg/dL vs. -0.12 ± 0.34 mg/dL, p = 0.012) compared to the NS group. Although a more pronounced decrease in urea levels was observed in the NAC group, this was not statistically significant. A reduction in creatinine levels of ≥25% was detected in 60.9% of patients in the NAC group, compared to 36% in the saline group (p = 0.027). Contrast-induced nephropathy (CIN) developed in 24% of the saline group patients, while no CIN cases were observed in the NAC group.

Discussion: High-dose NAC treatment in the ED appears to be an effective strategy for preventing CIN in patients with mild renal dysfunction (SCr levels of 1.2-1.8 mg/dL) who are scheduled to receive contrast media.

**Keywords**

N-Acetylcysteine, Contrast-Induced Nephropathy, Emergency Department, Prevention

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## Introduction

Acute kidney injury (AKI) that occurs shortly after the administration of intravenous (IV) iodinated contrast for radiological imaging is referred to as contrast-induced AKI (CI-AKI) or, more commonly today, contrast-induced nephropathy (CIN) [1]. Although these two pathophysiological definitions are debated as involving different mechanisms, both describe an iatrogenic clinical condition that results in nephron damage [2, 3]. Although nearly 70 years have passed since the first case of CIN was described, there is still ongoing debate about the best approach to combat this iatrogenic disease [4].

The potential of intravenously administered contrast media to cause CIN, particularly for diagnostic purposes, varies significantly depending on the presence of risk factors, the type of contrast used, the volume of contrast administered, the route of administration, and the patient population being studied. Consequently, while the incidence changes with the presence of risk factors, the incidence of CI-AKI is considered negligible in their absence. However, in patients with risk factors, the incidence of post-contrast AKI has been reported to range from 12% to 50% [5, 6].

Today, emergency departments (EDs) are among the most frequent and rapidly growing settings for the use of contrast media, particularly for diagnosing life-threatening conditions that require urgent and accurate diagnosis. Imaging with contrast is increasingly employed to facilitate timely decision-making in critically ill patients [7]. The rapid advancement of medical imaging technology, the frequent preference for minimally invasive imaging methods in diagnostic tests, and clinicians' reliance on contrast use in managing patients' diagnostic processes have contributed to CIN remaining a growing clinical issue. Therefore, developing strategies to combat this iatrogenic condition that arises during the diagnostic process is of great importance. When examining these strategies, efforts have been made to prevent this iatrogenic condition by targeting the pathophysiological mechanisms involved in CIN development. Approaches include the use of agents such as sodium bicarbonate, hydration with 0.9% NaCl solution- normal saline (NS), N-acetylcysteine (NAC), Vitamin C, Statins, Hemofiltration, and Hemodialysis, or Short-term controlled tissue ischemia, as well as medications like Metformin, angiotensin-converting enzyme inhibitors (ACEIs), and angiotensin II receptor blockers (ARBs), administered before or after contrast exposure [8]. Although NAC, a potent antioxidant, is believed to have a positive effect on renal hemodynamics by directly reducing renal oxidative damage, it remains a controversial topic due to inconsistencies in research findings [9]. For this reason, in our study, we aimed to evaluate the efficacy of high-dose NAC treatment in preventing CIN in the ED.

## Material and Methods

### *Study Design and Setting*

This study is a retrospective analysis of a randomized controlled trial (RCT) conducted as part of a dissertation thesis in the ED at Dr. Lütfi Kırdar Kartal City Hospital. The primary aim of the original RCT was to evaluate the efficacy of high-dose NAC compared to NS in preventing CIN in patients with mild renal impairment (serum creatinine [SCr] levels between 1.2 and 1.8 mg/dL). In this retrospective analysis, propensity score matching (PSM) was employed to balance baseline characteristics between the two treatment groups and to strengthen the comparability of the NAC and Salin groups, thereby improving the accuracy and validity of the estimated treatment effects.

### *Study Population*

Patients aged 16 years and older, with a baseline serum creatinine level of 1.2–1.8 mg/dL, who were included in the original RCT, were eligible for this analysis. Exclusion criteria included: known allergy to NAC or contrast agents, history of contrast-induced nephropathy, chronic heart failure (NYHA class III-IV), end-stage renal disease requiring dialysis, severe hepatic impairment, any active malignancy, inadequate laboratory data, or the need for urgent computed tomography (CT) imaging as determined by the treating physician. Out of the original RCT participants, 48 patients met the inclusion criteria for this retrospective analysis. These patients were randomized into two groups: the NAC group (n=23) and the NS group (n=25).

### *Randomization and Interventions*

In the original RCT, patients were assigned to treatment groups based on the order of hospital admission (odd numbers assigned to NAC, even numbers of toNS). The NAC group received 150 mg/kg of NAC intravenously in 0.9% NaCl-NS over 30-60 minutes before contrast administration (pre-Contrast), followed by a maintenance dose of 50 mg/kg over 2-4 hours post-Contrast [10]. The NS group received IV 0.9% NaCl at 6-8 mL/kg/h for 60-120 minutes before contrast and 3-4 mL/kg/h over 4-6 hours post-contrast. Low-osmolar iohexol (Omnipaque 350 mg/100 ml) was used as the contrast agent.

### *Data Collection and Variables*

For the retrospective analysis, baseline demographic data, including age, gender, and initial serum creatinine and urea levels, were collected. SCr and urea were measured at baseline, 24 hours, and 72 hours post-contrast administration. The primary outcome of this analysis was the change in creatinine and urea levels, defined as an increase or decrease from baseline to 24 or 72 hours. Secondary outcomes included the proportion of patients achieving a ≥25% reduction in SCr and urea levels and the development of CIN, defined as an increase in SCr of ≥0.5 mg/dL.

### *Propensity Score Matching and Analysis*

Propensity score matching (PSM) was performed to ensure balanced baseline characteristics between the NAC and NS groups in the retrospective analysis. Propensity scores were calculated using a logistic regression model based on age, baseline SCr, and baseline urea levels. Nearest neighbor matching without replacement was applied to match each patient in the NAC group with a control from the NS group. Balance was assessed using standardized mean differences (SMD), with an SMD <0.1 indicating good balance.

### *Analysis*

Comparative analyses between groups were conducted using Student's t-test or Mann-Whitney U test for continuous variables and chi-square or Fisher's exact test for categorical variables. Changes in creatinine and urea levels were analyzed using linear regression models, adjusting for baseline characteristics. Results were reported as mean differences with 95% confidence

intervals (CIs) using the format [mean difference (95% CI)]. Statistical significance was set at $p < 0.05$. Data analyses were conducted using Python.

*Ethical Approval*

This study was approved by the Ethics Committee of Kartal Dr. Lütfi Kırdar City Hospital (Date: 2014-12-09, No: 6).

## Results

After propensity score matching, 23 patients remained in each group. Baseline characteristics were well-balanced, with no significant differences in age (NAC: 64.4 ± 12.8 vs. NS: 61.8 ± 10.9, p = 0.453), baseline SCr (NAC: 1.48 ± 0.19 mg/dL vs. NS: 1.50 ± 0.18 mg/dL, p = 0.723), and baseline urea (NAC: 79.6 ± 51.2 mg/dL vs. NS: 87.9 ± 30.8 mg/dL, p = 0.523) (Table 1). The gender distribution was also similar, with 60.9% male in the NAC group and 65.2% male in the NS group (p = 0.784).

From baseline to 24 hours, the NAC group demonstrated a significantly greater reduction in SCr levels (-0.27 ± 0.15 mg/dL) compared to the NS group (-0.08 ± 0.29 mg/dL), with a mean difference of -0.19 [95% CI: -0.35, -0.03], p = 0.022 (Table 2).

Similarly, from baseline to 72 hours, the NAC group showed a larger reduction in SCr levels (-0.46 ± 0.22 mg/dL) compared to the NS group (-0.12 ± 0.34 mg/dL), with a mean difference of -0.34 [95% CI: -0.60, -0.09], p = 0.012 (Figure 1).

Urea levels also showed a significant decline in the NAC group. From baseline to 24 hours, the NAC group had a mean urea reduction of -13.13 ± 14.70 mg/dL compared to -11.13 ± 30.08 mg/dL in the NS group. Although this difference was not statistically significant, by 72 hours, the NAC group exhibited a more pronounced reduction in urea levels (-30.96 ± 32.83 mg/dL) compared to the NS group (-17.83 ± 19.80 mg/dL), with a mean difference of -13.13 [95% CI: -27.30, 1.04], p = 0.068 (Figure 2).

The NAC group continued to show a significant reduction in urea levels from 24 to 72 hours, with a mean decrease of -17.83 ± 23.69 mg/dL compared to -6.70 ± 20.11 mg/dL in the NS group, with a mean difference of -11.13 [95% CI: -23.51, 1.24], p = 0.078.

A significantly higher proportion of patients in the NAC group achieved a ≥25% reduction in SCr levels at 72 hours (60.9%) compared to the NS group (36%), with a difference of 24.9% [95% CI: 3.5, 46.3], p = 0.027. In terms of urea levels, 52.2% of patients in the NAC group had a ≥25% reduction at 72 hours compared to 34.8% in the NS group, although this difference was not statistically significant (p = 0.151).

Regression analysis indicated that NAC treatment was associated with a significantly greater reduction in SCr levels compared to NS, with a coefficient of -0.36 [95% CI: -0.64, -0.09], p = 0.014 (Table 3). Similarly, for urea reduction from baseline to 72 hours, NAC treatment showed a significant effect, with a coefficient of -17.57 [95% CI: -28.88, -6.26], p = 0.003. Baseline urea levels were also a significant predictor of the change in urea, with higher baseline levels associated with greater reductions (coefficient: -0.49 [95% CI: -0.64, -0.35], p < 0.001).

In terms of the development of CIN, no patients in the NAC group developed CIN at 24 or 72 hours. In contrast, six patients

**Table 1.** Baseline Characteristics of Matched Patients

| Variable | NAC Group (n=23) | Salin Group (n=23) | p |
|---|---|---|---|
| Age (years) | 64.4 ± 12.8 | 61.8 ± 10.9 | 0.453 |
| Baseline Creatinine (mg/dL) | 1.48 ± 0.19 | 1.50 ± 0.18 | 0.723 |
| Baseline Urea (mg/dL) | 79.6 ± 51.2 | 87.9 ± 30.8 | 0.523 |
| Male, n (%) | 14 (60.9%) | 15 (65.2%) | 0.784 |

**Table 2.** Changes in Creatinine and Urea Levels Post-Contrast

| Variable | NAC Group (n=23) | Salin Group (n=23) | p | Mean Difference (95% CI) |
|---|---|---|---|---|
| Δ Creatinine (Baseline to 24 hr) | -0.27 ± 0.15 | -0.08 ± 0.29 | 0.022 | -0.19 (-0.35, -0.03) |
| Δ Creatinine (Baseline to 72 hr) | -0.46 ± 0.22 | -0.12 ± 0.34 | 0.012 | -0.34 (-0.60, -0.09) |
| Δ Urea (Baseline to 24 hr) | -13.13 ± 14.70 | -11.13 ± 30.08 | 0.622 | -2.00 (-16.15, 12.15) |
| Δ Urea (Baseline to 72 hr) | -30.96 ± 32.83 | -17.83 ± 19.80 | 0.068 | -13.13 (-27.30, 1.04) |
| ≥25% Creatinine Reduction, n (%) | 14 (60.9%) | 9 (36%) | 0.027 | 24.9% (3.5, 46.3) |
| ≥25% Urea Reduction, n (%) | 12 (52.2%) | 8 (34.8%) | 0.151 | 17.4% (-7.8, 42.5) |

CI: Confidence Interval

**Table 3.** Regression Analysis for Creatinine and Urea Change

| Variable | Coefficient (β) | Standard Error | p |
|---|---|---|---|
| NAC (vs. Salin) - Creatinine | -0.36 | 0.14 | 0.014 |
| NAC (vs. Salin) - Urea | -17.57 | 5.60 | 0.003 |
| Age (years) | 0.01 | 0.01 | 0.210 |
| Baseline Creatinine (mg/dL) | -0.20 | 0.10 | 0.064 |
| Baseline Urea (mg/dL) | -0.49 | 0.07 | <0.001 |

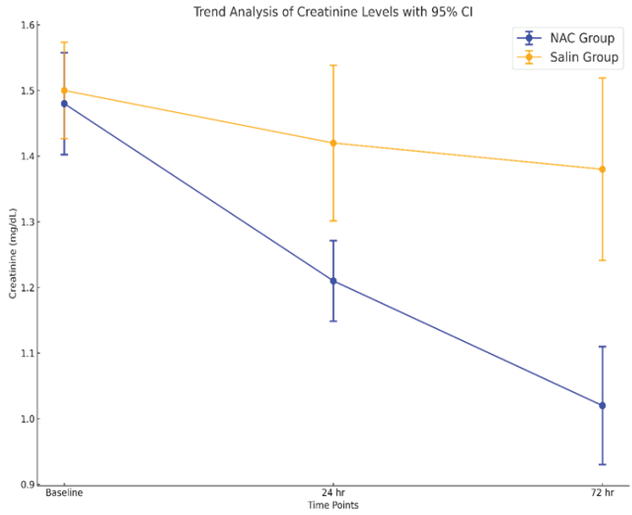


**Figure 1.** Trend analysis of creatinine levels in the NAC and Salin groups at baseline, 24 hours, and 72 hours post-contrast, with 95% confidence intervals.

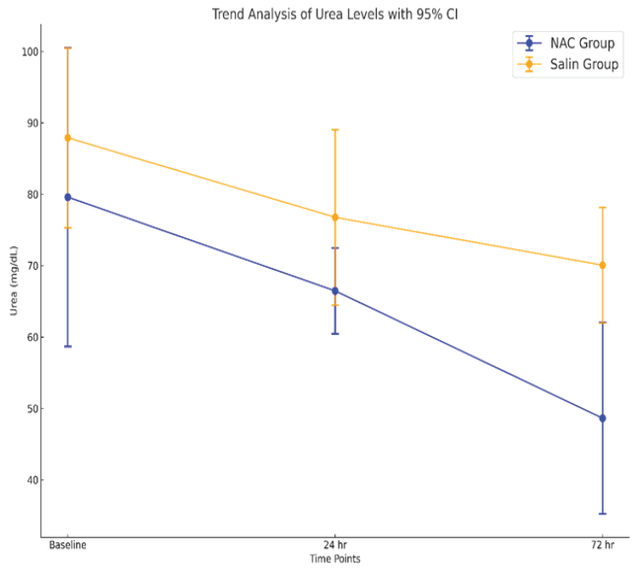


**Figure 2.** Trend analysis of urea levels in the NAC and Salin groups at baseline, 24 hours, and 72 hours post-contrast, with 95% confidence intervals.

in the NS group developed CIN at 24 hours (24.0%) and three patients at 72 hours (12.0%). The difference was statistically significant at 24 hours (p = 0.023) but not at 72 hours (p = 0.235).

## Discussion

In this study, the efficacy of NAC administered before (pre-contrast) and after (post-contrast) contrast exposure in preventing CIN was investigated in patients with mild renal dysfunction (SCr levels of 1.2-1.8 mg/dL), compared to NS given before and after contrast administration. PSM was used to balance the baseline characteristics between the NAC and NS groups, allowing for a more accurate assessment of treatment effects. More pronounced reductions in SCr and urea levels were observed in the NAC group compared to the NS group. While a significant reduction in SCr levels was detected at 24 and 72 hours in the NAC group, the decrease in the NS group was more limited. Although the NAC group showed more prominent reductions in urea levels, this difference was not statistically significant. The proportion of patients with ≥25% reduction in SCr e levels at 72 hours was significantly higher in the NAC group compared to the NS group. Additionally, no cases of CIN were observed in the NAC group, whereas a certain proportion of CIN development was noted in the NS group. High-dose NAC treatment appears to be effective in reducing the risk of CIN in patients with mild renal dysfunction.

Accurate measurement of kidney function is crucial for the routine care of patients and for determining the status of renal function. Glomerular filtration rate (GFR), considered the gold standard for monitoring kidney function, defines the rate at which fluid is filtered through the kidneys. The gold standard method for measuring GFR involves the injection of inulin, followed by the measurement of its clearance by the kidneys [11]. This method is invasive, time-consuming, and expensive. Therefore, as an alternative, SCr — a biochemical marker found in serum and urine— is the most used point-of-care test for both estimating GFR and monitoring kidney function. It is a rapid, cost-effective biomarker [12, 13]. Therefore, in this study, the definition of the high-risk group was based on SCr levels, with the lower threshold set at 1.2 mg/dL, following international laboratory recommendations, regardless of gender differences [14]. In our study, we observed that NAC administration reduced SCr levels. This observation is consistent with findings from other studies in the literature [15]. Although some studies argue that NAC's effect may be independent of a change in GFR, potentially increasing SCr tubular secretion or decreasing creatinine production, our study suggests a positive impact of NAC on SCr and, thus, on renal function. This is supported by the observation of lower SCr levels at both 24 and 72 hours compared to the NS group.

The exact mechanism leading to CIN following contrast administration has not yet been fully elucidated through prospective studies. The most likely mechanism remains the combination of contrast media-induced renal vasoconstriction, resulting in hypoxia, along with direct toxicity to the tubular epithelial cells, which together impair kidney function [16]. As a result, reactive oxygen species produced in connection with contrast media administration are considered to play a pivotal role in the development of CIN [17]. Because reactive oxygen species can affect both cortical and medullary microcirculation directly and indirectly, leading to vasoconstriction, antidiuresis, and anti-natriuresis, superoxide dismutase, a scavenger of reactive oxygen species, may inhibit the kidney damage caused by contrast media [18]. Therefore, NAC, which acts on this pathway, remains one of the most frequently studied agents for preventing CIN. The European Renal Best Practice (ERBP) position statement on the Kidney Disease Improving Global Outcomes (KDIGO) Clinical Practice Guidelines on Acute Kidney Injury recommends the use of oral NAC only in patients receiving appropriate fluid and salt loading (2D). It also advises against using oral NAC as the sole method for preventing CIN [15]. In this study, all patients were administered serum saline both before and after contrast media administration. While this intervention influenced SCr levels, the difference became more pronounced in patients receiving both NS and NAC at 24 hours,

and this effect was even more evident at 72 hours.

In studies conducted in the ED, Turedi et al. reported a high CIN incidence of 23.7% despite the use of at least one prophylactic measure for all ED patients before and after contrast administration [19]. In their study, the prophylactic measures included 3 mL/kg IV NAC + NS (3 g NAC diluted to 1000 mL with NS) or NaHCO3 + NS solution, or at least 1 mL/kg IV NS administered for a minimum of 6 hours after contrast administration. In comparison to our study, this study administered lower amounts of NS and NAC. Overall, significant variations in CIN incidence among ED patients have been reported, which may be attributed to various factors such as underlying patient conditions and the use of different CIN definitions.

In summary, this study divided patients with mild renal dysfunction (SCr levels of 1.2-1.8 mg/dL) into two groups: one receiving only NS before and after contrast administration and the other receiving both NS and high-dose NAC. The NAC group showed significantly greater reductions in SCr and urea levels compared to the NS group. At 72 hours, 60.9% of patients in the NAC group experienced a ≥25% reduction in SCr levels, compared to 36% in the NS group. Furthermore, none of the patients in the NAC group developed CIN, whereas CIN occurred in 24% of the NS group.

*Limitation*

This study has several limitations: 1) Although the data were derived from a randomized controlled trial, the retrospective nature of this analysis may introduce inherent biases that could affect the accuracy and reliability of the findings. Despite the use of propensity score matching to balance baseline characteristics, unknown confounding variables may still be present. 2) The study included a relatively small number of patients. This limited sample size may reduce the statistical power of the analysis and restrict the generalizability of the results to broader populations. 3) This study was conducted in a single tertiary hospital's ED, which may limit the generalizability of the findings to other healthcare settings or populations with different characteristics.4) The study assessed outcomes within 72 hours post-contrast administration, but no long-term follow-up was conducted. Long-term kidney function and the potential for delayed contrast-induced nephropathy were not evaluated. 5)The study focused on patients with mild renal impairment (SCr levels between 1.2 and 1.8 mg/dL). Patients with more severe renal dysfunction, who may be at higher risk of CIN, were excluded, which could limit the applicability of the findings to higher-risk populations. Future studies comparing multiple agents could provide a more comprehensive assessment of CIN prevention strategies.

*Conclusion*

In the ED setting, for patients undergoing CT with low-osmolar contrast media, high-dose IV NAC treatment (150 mg/kg NAC in 0.9% NaCl administered 30-60 minutes before contrast, followed by a maintenance dose of 50 mg/kg within 2-4 hours after contrast administration) stands out as an effective option for preventing contrast-induced nephropathy in patients with mild renal dysfunction (SCr levels of 1.2-1.8 mg/dL).

***Scientific Responsibility Statement***

*The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.*

***Animal and Human Rights Statement***

*All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or compareable ethical standards.*

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***Conflict of Interest***

*The authors declare that there is no conflict of interest.*

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